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Short Communication :

Renal Ultrastructural Alterations in Ketone-Induced Potentiation of Chloroform Toxicity

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Chloroform ($CHCl_3$), a chlorinated hydrocarbon is potentially hepato - and nephrotoxic that required bioactivation to the proximate toxin via metabolism by cytochrome P-450 mixed-function oxidases (MFO) resulting in covalent binding to macromolecules. Ketonic solvent such as 2-hexanone (2-Hx) may potentiate the hepatotoxicity and nephrotoxicity of $CHCl_3$. Many investigators recently reported the possibility that renal mitochondria may be the site of halocarbon ($CHCl_3$, CCl_4) biotransformation rather than in the endoplasmic reticulum the site of biotransformation in the liver.

The following experiment was aimed to investigate the primary site(s) of $CHCl_3$ - ınduced nephrotoxicity as potentiated by -Hx, and a time sequence was applied to the experiments to define any differences in the onset and magnitude of the kidney injury in rats pretreated with corn oil (CO) or 2-Hx, a ketone, and then challenged with $CHCl_3$, the haloalkane. Morphologic and functional data

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from the present study support the possibility that $CHCl_3$ is biotransformed by the cytochrome P-450 MFO in the kidney mitochondria rather than in the endoplasmic reticulum, the bioactivation site of $CHCl_3$ in the liver. The new and interesting findings were the renal corpuscular lesions that may signify alteration of glomerular permeability induced by 2-Hx or $CHCl_3$ nephrotoxicity. Rats pretreated with 2-Hx alone and those that had 2-Hx/$CHCl_3$ treatment developed glomerular injury with numerous hyaline droplets in the S_1 and S_2 epithelial cells, which suggest an intense protein reabsorption due to increased glomerular permeability. Rats with CO/$CHCl_3$ regime of treatment developed renal corpuscular and proximal epithelial lesions followed with tubular epithelial regeneration at 6 and 48 hours after $CHCl_3$ exposure respectively while those with 2-Hx pretreatment never exhibit any tubular epithelial regeneration. Nephrotoxicity caused by 2-Hx, $CHCl_3$ or 2-Hx/$CHCl_3$ may be due to the toxic effect of the bioactivation of these compounds in the proximal epithelial cells resulting in damage to the renal tubular epithelium and renal corpuscles.